THE VETERINARY RECORD

A Weekly Journal for the Profession.

Edited by WILLIAM HUNTING, F.R.C.V.S.

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1903.

The year that has passed is to veterinarians perhaps most notable as the one in which our colleagues in the Army succeeded in obtaining rank on level terms with other of His Majesty's officers. Whatever complaint may be made of some of the details of the last Warrant it is, taken as a whole, a very distinct mark of progress for the A.V.D. and for the veterinary profession.

Another prominent feature of the year has been the remarkably unanimous wish expressed for a University Veterinary Degree. The holding of such a degree would mark a man as the possessor of something more than veterinary training. Many men with a veterinary diploma now possess some literary and artistic as well as scientific culture, but the letters M.R.C.V.S., are no more guarantee of the fact than are those of M.D. or M.R.C.S. We desire to obtain a University label which will frank us into a higher social circle. Whether this ambition is not a little premature we cannot yet say, but it will probably be capable of realisation during 1904. In Ireland, Scotland, and England, University authorities seem willing to arrange for a veterinary degree, and we hope that when the prize is offered we shall have candidates willing and eager to present themselves. Success means time and labour and money.

Our Civil Veterinary Department has done nothing worthy of special notice, unless it be a rearrangement of some of their methods of controlling swine fever. This disease shows just such a decline in prevalence as to suggest what might be done by a little more boldness of action. Tuberculosis is still outside of the schedule of diseases thought by the Board of Agriculture sufficient for the protection of the stockowner. Glanders might as well be outside also for all the good existing regulations are. Anthrax and sheep-scab are both increasing, despite the fact that regulations for their control have been in force for some years.

1903 has been a year of remarkable health among stock. Both in town and country epizootics (save those looked after by the Board of Agriculture) have been almost unknown. Influenza and strangles have caused less trouble than for years past. This is very satisfactory for stockowners, and veterinarians are not selfish enough to wish any increase of disease. They only say—call us in early and you will find that economy attends our visits.

We wish all our readers a happy and prosperous New Year.

OSTEOMALACIA ASSOCIATED WITH PERIOSTITIS.

By JOHN B. WOLSTENHOLME, F.R.C.V.S., Manchester.

Bay gelding, hunter, over 20 years of age. Writer had known him for some three years, as the property of a surgeon who used him for harness and hack work. During this period he did not carry much flesh, but worked well and with spirit. Had been sent to grass in the early part of the summer, and had returned to the stable in good condition considering the age and general habit.

One morning he was found in the loose box with marked stiffness and rigidity about the neck and spine generally, for which there was no cause ascertainable. He continued to eat and drink in the box as usual.

It was on July 25th I first saw him, which was about one week from the commencement of symptoms. At this time he was in very poor condition, pulse and temperature normal, appetite and secretions apparently undisturbed. There was marked rigidity of the spine when seen at rest or in movement. He was afraid of anyone approaching him, and would move backward with a slight groan when that was attempted. A turning movement to right or left was made with great care, and rigidity of the entire spine, as though in pain.

On manipulation of the neck some little swelling and heat were noticed at the lower part, over the vertebræ. The horse could raise and depress his head, or suffer it to be done, without much distress; but the slightest lateral movement caused acute pain, which was evinced by groaning and stepping backwards. On closer and careful handling an imperfect crepitus could be detected, which led to the diagnosis that fracture of one or more of the lateral processes was present.

The owner was advised to have the horse destroyed, which was done after a delay of about a week.

Post-mortem. No enlargement of the facial or cranial bones. The joints and limbs were perfectly "fine." On cleaning off the muscular tissue from the cervical vertebræ there was considerable congestion found in the parts close to the bones, together wi h fracture of the right transverse process of the sixth and of the seventh. The lower cervical vertebræ appeared to be hyperæmic, and were readily cut into with the knife.

There was no tumour nor new growth in the neck or other part of the body. No caseation nor deposit that suggested tubercle. There was an exostosis of

about 3 by 2 inches beneath the anterior dorsal vertebræ, and two smaller ones beneath the posterior, they were of old standing, and did not yield to the knife, nor was any other part of the skeleton noticed so to respond.

The bones of the neck were boiled at the knacker's, and when returned were as follows:—

Atlas. Some periosteal deposit on the left pedicle, also above and below left wing. Some erosion of upper surface of body, (*i.e.*, floor of spinal canal) on left half. Weight not much altered.

Dentata. Body thickened up to anterior articular surfaces by deposit of porous bone.

Odontoid process normal. Deposit about arterine foramen in each transverse process.

Third. This bone was damaged by a dog; it had, however, much of the appearance of the fourth.

Fourth. Body much thickened with very porous bone, which latter forms a collar around the articular process at one extremity, and the articular concavity at the other. Deposit on the pedicles. Slight deposit on outer surface of left superior oblique, and on anterior point of left transverse process. Bone lighter than normal.

Fifth. This has been photographed. Body *very much* thickened by deposition of spongy bone; it is rugose and furrowed. Both superior oblique processes affected. Bone lighter than normal.

Sixth. Body affected but not to the same extent as those of the 4th and 5th. Pedicles slightly affected. Both oblique processes on the left, and posterior oblique on the right affected, as also both transverse, with fracture of the posterior cusp of the right one. Bone lighter than normal, but not to the same extent as the two last mentioned.

Seventh. Body very thickened and rugose. Pedicles, laminæ and oblique processes affected. Superior spinous process slightly diseased. Right transverse process fractured. Bone fairly heavy.

In this case it is to be noticed that *no arthritis* had existed, although the morbid process had extended right up to the articular surfaces.

There was no "big-head" as commonly occurs in osteo-porosis affecting the horse.

In *The Veterinary Record* of April 11th, 1903, there is a reprint from the American Veterinary Review, on "Spinal Rigidity in the Horse," by W. L. Williams, V.S., of the New York State Veterinary College, Ithaca, N.Y., in which a case similar to the above is described under the heading of Osteo-Myelitis, etc., as occurring in an old mare.

In *The Journal of Comparative Pathology* for 1899, page 300, Harold B. Elliott, M.R.C.V.S., of the Island of Hawaii, H.I., gives many interesting particulars in connection with osteo-porosis, which may or may not have some relationship with osteomalacia.

Friedberger and Fröhner, in their pathology, give the best account of osteomalacia which I have seen.

It would possibly be well to mention that I have noticed that the ribs and other bones of the trunk often cut easily with the knife when making post mortems on Iceland ponies, especially when aged, and those which had a peculiar stiff waddling gait during life.

THE TREATMENT OF FISTULOUS WITHERS IN AUSTRALIA BY BUSH DOCTORS.

In this country horses are cheap, comparatively speaking, and there is a plethora of people who consider they are capable of tackling any sort of case that crops up—provided the animal belongs to a neighbour and they are not directly financially interested therein. Probably the most common trouble met with is fistulous withers, and the following have come under my personal notice quite recently and may be taken as fair examples.

Case I. Bay gelding, 6 years old, 15·2, broken to harness, worth about £20. Owner noticed swelling on wither when horse was running at grass. Swelling hard. Blistered—softened and broke. Swelling now reduced, but constant discharge from two openings. Mixture composed of sulphate of copper and stale urine (human) injected into openings for past two months. This prescription was given after a little pressure as a great personal favour.

Case II. Milk cart mare, 4 years old, worth about £18. History: mare developed enormous swelling on each side of neck immediately in front of wither, which, after bathing with hot water and blistering, broke. Swelling not much reduced, discharging from three openings. When seen by me she was under treatment by a man with a great reputation. The treatment, as far as I could understand it, consisted in passing two large setons right through the neck and attaching lead discs about 6ozs. in weight on each side. The setons were made of hairs from the tail, and would measure ½in. in diamter. In addition to this treatment a powder was passed into the centre of the swelling. I was informed that this powder was a secret remedy for which one of Melbourne's leading surgeons had offered the sum of £50, but the quack would not part! When I saw the case a swelling resembling a football was sloughing out *bringing the skin with it:* the process had taken 2½ months, and was a long way from being complete. I saw evidence of the terrible, deep, caustic action, and believe Hyd. Perchlor, and Cupri Sulph. were used as a powder. I was informed that the mare had fallen away very much, in fact for two weeks after the setoning and powdering they had a hard task to get her to eat. I was asked to operate, but prefer to wait and see how much horse is left when the slough comes away,

Hyd. Perchlor. wrapt in tissue-paper and probed to the bottom of the fistula is a favourable and often successful bush remedy (up country remedy.) Another one which is, I am glad to say, going out of use is to hang a piece of meat until it teems with "jumpers," and then scoop them into the wounds on the wither. The general belief is that the maggots live on and consume the diseased tissue only.

I do not know why fistulous withers should be so common out in this country, but they are, and what is more they are nearly always associated with, if not caused by, fracture of one or more spinous processes of the vertebræ.

ROVER.

ABSTRACTS FROM FOREIGN JOURNALS.

INVESTIGATIONS IN THE DIARRHŒA OF CALVES.—By Dr. JOEST, of Kiel, Veterinary Surgeon.

The term "calf scour" does not denote a uniform specific infectious illness, but it is a collective name for several aetiologically different affections.

Joest tried to differentiate these, and dissected and studied 23 subjects, part of which were slaughtered and part of which died naturally. These subjects were dispatched promptly to the bacteriological institute at Stettin out of 21 different outbreaks in Pomerania. The pathological anatomical state showed "calf scour" in 20 cases. In each one there was hæmorrhagic enteritis, polyarthritis or calf lameness, and catarrhal pneumonia. Both the latter, the author, after an exhaustive bacteriological examination, considers to be due to a mixed infection with coli-bacteria; the enteritis he looks on as probably due to a toxic diarrhœa of calves. In 20 other calves the principal changes were catarrhal inflammatory appearances in the stomach and intestine, inflammatory swelling of the lymphatic glands of these parts, parenchymatous degeneration of the liver, kidneys, heart muscle, hæmorrhages under the entire serous membranes and the endocardium—all the usual appearances of septicæmic illnesses in general, nothing pathognomic. Bacteriological examination revealed in all cases the same bacteria present on the peritoneum, and in the organs of the abdominal cavity in greatest number as well as in the heart blood. These in their morphological and biological character resembled the bacterium coli, and were called by the author the bacteria of calf scour. They are the same as those found by Jensen and Poels, and identical after inoculation results.

Joest inoculated small animals with the isolated bacteria, bred in suitable media. Guinea-pigs soon died after intra peritoneal inoculation; they scarcely reacted at all on subcutaneous inoculation. White mice died on subcutaneous and peritoneal inoculation. Rabbits were killed with fairly large doses injected intravenously.

In experiments on calves, new born healthy calves when given the cultures incorporated with milk were affected clinically and pathologically with a qualified calf scour illness. Calves whose stomach and intestine were already functionally active through the taking of nourishment are little affected, proving the important prophylactic fact that the partaking of mother's milk immediately after birth counteracts the occurring of calf scour in the digestive tract.

As the port of entrance of infection, besides the digestive tract, the navel must be considered. An infection of the navel will kill animals 12 hours old with a typical calf scour, and there will be thrombosis of the vessels of the navel. Intravenous introduction of a small amount of culture of the bacteria killed calves, and they showed most severe appearances of a general infection.

In both the latter kinds of infection the severe intestinal lesions may be regarded as secondary, and the diarrhœa is not directly due to a diseased intestine but to a septicæmia, which indeed for the most part is caused by infection of the intestine, for fasting animals and animals with a small power of resistance possess intestinal mucous membranes, especially prone to infection.

The serum acquired for agglutination operations only caused a temporary or insignificant diarrhœic affection. The serum of rabbits, not treated before, agglutinised calf scour coli-bacteria in dilutions of 1:5—10. The serum, with a fixed coli-stock, of rabbits previously treated, agglutinised even this stock very strongly 1:500—1000. On the contrary several other races of coli possessed no higher agglutination value than the customary bacterium coli.

A differential diagnostic means of distinguishing the usual coli bacteria from the coli-bacteria of calf scour has not been made out.

The serum of a rabbit immunised with a calf scour coli stock in a sernm culture mixture experiment on a guinea pig has a protective effect against a certain deadly dose of the stock used for the immunisation in the quantity of 0·1 c.cm. On the contrary it does not protect against several other species of coli-bacteria.

For the practical object of immunisation several different kinds of calf scour coli-bacteria must be brought to our aid in experiments, and a polyvalent calf scour serum produced analogous to the polyvalent serum of swine plague.

The author is still busy with prophylactic and therapeutic employment of the serum, and will report results in a later publication.—*Ex Berliner Thierärzt Woch.*

G. M.

Royal College of Veterinary Surgeons.

EXAMINATIONS IN LONDON.

At meetings of the Board of Examiners held in London, on December 11th for the Written, and on and between 15th and 16th for the Oral and Practical Examinations, the following gentlemen passed their final examination.

Mr. B. H. Benson
J. C. Broad
J. McRae Frost
R. C. Glover
T. S. Hunter *
R. Hayes
J. Hobday
R. P. Johns
Mr. T. Love
A. R. Lewis
T. A. Nicholas
H. K. Roberts
J. Richardson
F. R. Staples
D. O. Turnbull
W. H. Walker *

The following passed their Third Examination:—

Mr. A. C. Anderson
R. S. Audas *
G. G. Brown
P. J. Bolton
J. A. Bosley
L. L. Dixson
Mr. F. Hewson
E. G. Haskell
W. J. Macauley
J. M. Richardson
W. R. Townson

The following passed their Second Examination :—

Mr. T. Bell	Mr. E. R. Nichols
H. Rrassey	P. Stanley
L. L. Balls	G. C. H. Sharpe
P. Carter	H. G. Stewart
A. G. Doherty *	C. P. Wellington
J. H. Jones	J. B. Welham
R. W. Mellard *	R. C. Wilkinson

The folowing passed their First Examination :—

Mr. A. Akin Higgins	Mr. R. Gooderidge
L. B. Cole	B. A. Jarvis *
A. F. Castle	E. F. Land

Mr. A. J. S. Reynolds.

Those marked thus * passed with 2nd Class Honours.

ARTHUR WM. HILL, *Secretary.*

EXAMINATIONS IN EDINBURGH AND GLASGOW.

At meetings of the Board of Examiners held in Edinburgh and in Glasgow on December 11th for the Written Examination, and on and between December 17th and 21st for the Orals, the following gentlemen passed their Final Examination and were admitted Members of the Royal College of Veterinary Surgeons :—

DICK COLLEGE.

Mr. A. J. Horner	Mr. W. F. Thomas
W. Nairn	

NEW COLLEGE.

Mr. R. W. Jones	Mr. J. C. Smith
W. D. Lindsay	F. R. Shippard

GLASGOW COLLEGE.

Mr. P. F. Tubridy	Mr. R. Bickerton
K. McL. McKenzie	H. McIntyre

DUBLIN COLLEGE.

Mr. W. J. B. Devine.

The following passed the Class C or third examination :—

DICK COLLEGE.

Mr. D. Hutch	Mr. A. A. White
W. W. Kerr	W. F. McConnell
J. W. Rainey *	J. Donaldson

NEW COLLEGE.

Mr. W. G. Darling	Mr. J. O'Leary
E. Elphick	

The following passed the Class B or second examination :—

DICK COLLEGE.

Mr. A. T. Crowther	Mr. G. W. Rutherford
J. Dods	T. W. Smith
W. K. Johnstone	W. J. Young
A. H. Leyland	

NEW COLLEGE.

Mr. H. R. Laycock	Mr. P. J. Walsh
W. Walker	R. T. Wood

GLASGOW COLLEGE.

Mr. W. Holliday	Mr W. Hill
W. D. Blair	

The following passed the Class A or first examination :—

DICK COLLEGE.

Mr. H. S. A. Dunn *	Mr. L. A. Ramsay
E. C. Howard	P. T. Saunders
O. Jones	R. Waddy *
P. B. Noble	

NEW COLLEGE.

Mr. T. G. Shand	Mr. J. C. Storie
W. Simister	W. P. Ruthven

GLASGOW COLLEGE.

Mr. P. McCorry	Mr. R. Hopps
W. J. Philips *	W. Henderson

Those marked thus * passed with second class honours.

R. RUTHERFORD, F.R.C.V.S.

Secretary in Scotland to the Board of Examiners.

THE CENTRAL VETERINARY MEDICAL SOCIETY.

An ordinary monthly meeting was held at the Royal College of Veterinary Surgeons, 10 Red Lion Square, W.C., on Thursday, December 3rd. Mr. R. Porch (President) in the chair. There were present: Messrs. A. L. Butters, J. Rowe, F. G. Samson, S. H. Nye, R. A. Philp, W. Perryman, C. H. Sheather, A. R. Routledge, J. B. Hare, R. J. Foreman, H. A. MacCormack, A. Gill, W. R. Clarke, E E. Martin, C. E. Wells, P. S. Howard, J. A. Nunn, W. H. Flook, P. W. D. Smith, W. S. Mulvey, H. King, J. A. Gosling, L. Savournin, G. D. Martin, J. S. Hurndall, W. Pritchard, G. H. Broad, W. Hunting, H. Gray, G. Sutton, W. Macdonald, C. W. W. Brown, F. Hobday, T. Wolsey, and E. Lionel Stroud, Hon. Sec., and twelve visitors.

The minutes of the previous meeting were read and confirmed.

CORRESPONDENCE.

The SECRETARY read a letter from the South Durham and North Yorkshire Veterinary Medical Society, acknowledging his letter, and saying that they had taken similar action as the Central Veterinary Medical Society with regard to the Army Veterinary Department.

A letter was also read from the Victoria Veterinary Benevolent Fund asking the Secretary when posting letters to members to kindly enclose a leaflet of the Fund drawing attention to its claims.

On the motion of Mr. Howard, seconded by Capt. Martin, the request was acceded to.

A letter of resignation was received from Mr. W. E. Taylor, who now lived too far away from London to continue his membership.

On the proposition of Mr. Butters, seconded by Mr. MacCormack, the resignation was accepted with regret.

A letter was also read from Mr. Bailey asking to be placed on the list of country members.

SPECIMENS.

Capt. E. E. MARTIN exhibited some hock specimens. In the first case the animal had the characteristic lameness of spavin, but showed no external enlargement. The lameness lasted for about four months and gradually became worse. The case was diagnosed from the first as hock, and after the usual treatment of blisters and firing, no improvement being noticed, the animal was killed. The specimen was interesting because there was no enlargement visible externally; there was no exostosis of the bone at all, but when the bones were separated it was found that every joint was seriously involved. It showed what a large amount of articular disease might exist without any exostosis.

The second case was of a similar kind, with the cuneiform parvum bifid. He had taken some statistics of

OSTEOMALACIA. FIFTH CERVICAL VERTEBRA.
To illustrate Mr. Wolstenholme's case, p. 412.

CONTAGIOUS PNEUMONIA. SECTION OF LUNG SHEWING CRYSTALS.
To illustrate Prof. Henry Taylor's paper, p. 419.

the hocks at Aldershot, and out of sixty collected chiefly for spavin he found only three with the cuneiform parvum bifid.

Capt. Martin also exhibited a case of broken back which presented some features a little out of the common. The bones involved were the last bone of the dorsal vertebræ and the first of the lumbar. At the last manœuvres the rider of the horse in marching out from the camp suddenly found the animal staggering under him. He dismounted, but seeing nothing the matter, got on again, and the horse staggered along, but fell again. He was then placed on a gate and dragged into a field near by and lay there for four days. The veterinary surgeon of the column had gone on in front and the horse was treated by a civilian practitioner. That happened on the Monday morning, and the practitioner saw it on the Monday afternoon and again on the Wednesday, and reported paralysis. He (Capt. Martin) was sent up in a hurry and had no opportunity of consulting with the civilian. He found the horse lying in the field perfectly well in itself and flicking away the flies from its hind quarters with its tail. It had perfect control of its urinary organs, and also from time to time passed its dung. It fed fairly well, and got on its fore legs, but could not move the hind quarters, but there was sensation over all parts of the hind quarters. He had the horse destroyed and made a post-mortem, and on running his hand along the backbone found a projection of bone in the loins. The last bone of the dorsal vertebræ had broken away and the spine of the first lumbar vertebræ. It was an interesting case, showing how few symptoms there were of a well-marked fracture of the back.

Mr. FOREMAN exhibited a primitive kind of horse shoe which he had taken off a horse, as an illustration of what an animal could wear without going lame.

Mr. A. ROUTLEDGE exhibited specimens. On the previous day while in the knacker yard at Stratford he noted the peculiarly characteristic shape of the off hind foot of a bay horse brought in from the country by one of the carts. Unfortunately he had no history of the case, which was a very advanced case of pyramidal disease, showing the "buttress" formation, and in addition disease of the upper edge of the navicular bone. His friend, Mr. Willis, had given a very good account of that disease in *The Journal of Comparative Pathology* in June of this year, and from observations of a number of cases he was able entirely to agree with Mr. Willis in his remarks.

The second specimen was the inner hind foot of a black gelding, which, by means of posterior tibial neurectomy, was able to work soundly for three years, dying of another disease, On examination of the fractured pyramid it would be found to be loose, a sort of false joint being formed. That, he thought, was the rule, no bony union having taken place in any specimen he had examined.

The third specimen was the off fore foot of a chestnut mare, which after the median and ulnar operations worked thirteen months, finally being killed for rupture of the flexor pedis perforans tendon. The fourth specimen was probably the most interesting, as although there was a very extensive disease process going on in the pyramid, which was fractured and showed the false joint very well, the mare was never lame. She was killed as worn out and slow, and he made a post-mortem examination of the foot, which had had a buttress running down the front for two years. He also exhibited specimens of a bowel from a five-year-old horse which died of colicky symptoms lasting for forty-eight hours.

Mr. W. H. FLOOK exhibited various specimens of an ingenious little instrument for administering capsules to horses and dogs. The instrument was a long narrow tube which could be inserted in the animal's throat, and the capsule at the one end was then pushed out by means of a piston at the other.

Lieut.-Col. J. A. NUNN showed a calculus he had received from Capt. Barry, the veterinary officer of the 2nd Life Guards. The animal from which it was obtained was a black mare, a trooper of the 2nd Life Guards. She was nine years old. On 1st February, 1900, she suffered from an attack of colic and was on the sick list for four days. She did her work up to the 14th November last, when during the night she somewhat injured herself in the stable. She was treated, but during the whole time of treatment there was no passage through the bowels. The animal showed no pain, or even uneasiness, up to the 21st November, when she was suddenly attacked with violent pain and died in a few hours. The calculus was lodged in the sigmoid flexure of the colon. The calculus, which was about the size of an ordinary cocoanut, had a nucleus, so that there were two distinct calculi with no adhesion between them. There might be an explanation for the formation by considering that the kernel was always present in the bowel, but that it shifted its position in the attack of colic and then the second coating formed round it, and it again shifted its position, producing obstruction and death.

Prof. PRITCHARD, referring to the hock and the symptoms of the lameness, asked whether there was any indication that it was a case of articular disease. Everyone knew that with ordinary hock lameness and spavin there was an indication which could not be mistaken.

Capt. MARTIN said the only symptoms of the lameness was that the animal was very lame. It went on its toe, but when trotted for some little time it improved. In turning it over in its stall it showed great signs of lameness and stood with the hocks flexed. The only sign that could be found on manipulation was that the hock was a little bit hotter than the other at times, but the animal was blistered and fired, so that it was not always possible to tell whether the heat came from the disease or from the treatment.

Prof. PRITCHARD said he had some reason for asking the question. His experience told him that if there was lameness and ordinary bone spavin the longer the animal went the better it would go, but with spavin lameness from articular disease the further the animal went the worse he went, and that was a very important factor in connection with diagnosing cases of hock disease. As Captain Martin had pointed out, it was possible to have cases without any enlargement on the outside—he had known hundreds of them. With a case of hock lameness dragging the toe and other symptoms indicating hock disease, if the lameness did not lessen on progress it might be fairly concluded that the mischief was between the joints and not on the outside.

Capt. MARTIN said he had the animal trotted several times and it did improve. In working if it was rested it became very lame.

Lieut.-Col. NUNN, referring to Mr. Flook's instrument, considered that it was a modification of the balling gun and a useful instrument in dog practice.

Mr. SAMSON thought the instrument, like a good many others, was of very little use, and believed that if an animal put his teeth upon it it would become so damaged as to be useless.

Mr. FLOOK pointed out that the instrument was made of brass and would stand considerable biting. Very few coachmen could give balls nowadays without a balling gun, and the instrument would be a very useful one for coachmen and others having charge of animals.

Prof. PRITCHARD, referring to the calculus, asked for some information as to its character. Would he be wrong if he said it was not a calculus at all.

Lieut.-Col. NUNN said no; probably it was a concretion.

Prof. PRITCHARD said he meant an oat hair ball.

Lieut.-Col. NUNN said that was probably what it was.

ELECTION AND NOMINATIONS.

The following gentlemen were ballotted for and duly elected Fellows of the Society: Mr. JAS. DOUGLAS, 5, Regents Park Road; Mr. RICHARD ELLIOTT, of Bromley; and Mr. WILLIAM JOHN HATTON, Richmond, Surrey.

The following gentlemen were nominated for membership: Mr. J. WILLETT, of 6, Harley Place, Harley Street, W.; and Mr. A. E. SANGSTER, of 141, Finchley Road.

ON THE ETIOLOGY AND SOME OF THE PHASES OF CANINE DISTEMPER.—By HENRY GRAY, M.R.C.V.S.

(This paper appeared in *The Veterinary Record* of December 19th.)

DISCUSSION.

Lieut.-Col. NUNN said the paper was a most interesting one, and showed a vast amount of research. He desired to know if any experiments had been made with the potato cultivation of the bacillus, which was said to grow on the potato with difficulty. It would be interesting to know whether the bacillus cultivated on the potato retained its ordinary virulency or was decreased. He also wished the author's opinion as to the influence of climate and temperature on the course of the disease and the influence of the life which the animal led, such as dogs kept in houses, hounds in packs, shepherds' dogs, and so on. Then he would like to hear something as to the virulency and the course of the disease, and if there was any difference in foreign dogs which came from other climates and the native home-bred dog.

Capt. MARTIN said that with regard to the cutaneous eruption he had seen a case only a few weeks ago when a young dog was brought to him by the owner, who said he was looking a bit dull and had an eruption on the abdomen. He found the whole abdomen smothered with the cutaneous eruption of distemper, and told the owner to keep the dog quiet and warm, and let him know if it became any worse. Three or four days afterwards the dog had quite recovered. It was a question whether there was any relation between the quantity of eruption and the severity of the disease. It seemed to him the disease had expended itself in the eruption and had passed off with it.

Mr. W. HUNTING suggested that the discussion should be adjourned until the paper was seen in print, as it was a very complicated subject and could not be discussed thoroughly without being studied more carefully.

Prof. PRITCHARD seconded the motion, which was carried, and the discussion was adjourned, Mr. Gray promising to be present at the next meeting.

On the proposition of Prof. Pritchard, seconded by Mr. Butters, a vote of thanks was accorded to the Chairman.

On the motion of Mr. Butters, seconded by the Hon. Secretary, a cordial vote of thanks was given to the members who had exhibited morbid specimens, the Hon. Secretary pointing out that last year the Society had more morbid specimens than in any previous year of its existence, but he hoped that record was going to be beaten in the present year.

NORTH OF ENGLAND VETERINARY MEDICAL ASSOCIATION.

The quarterly meeting was held at the Douglas Hotel, Newcastle-on-Tyne, on Friday, Nov. 20th, 1903. The following members were present: Messrs. G. Craik, Alnwick (President); W. Hunter, J. Davidson, and H. S. Elphick, Newcastle-on-Tyne; J. Aitken, Chester-le-Street; T. Kilburn, Warkworth; F. Pickering, Hexham; G. R. Dudgeon, and G. M. Mitchell, Sunderland; A. Gofton, South Shields; E. R. Gibson, Seaham Harbour (hon. sec.) Visitors: Prof. Taylor, Edinburgh; Capt. McDougall, and Mr. Rankin, Newcastle.

The SECRETARY read the minutes of previous meeting, which were accepted.

Mr. McGregor, of Bedlington, wrote resigning his membership of the Association; the same was accepted with regret.

On the proposition of Mr. Dudgeon, seconded by Mr. Hunter, it was agreed that in view of Mr. McGregor's long connection with the North of England Association he having been one of the original members, that he be made an honorary associate.

A letter was read from the Veterinary Association of Ireland *re* service on juries. The Secretary was requested to acknowledge the receipt of same.

ELECTION OF OFFICERS.

President.—Mr. W. Hunter, Newcastle.
Vice-Presidents.—Messrs. Davidson and Aitken.
Auditors.—Messrs. Hunter and Davidson.
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OBSERVATIONS ON PASTEURELLOSIS OF THE HORSE.

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Mr. President and Gentlemen,—My first duty is to thank you for doing me the honour of inviting me to read a paper at your meeting to-day. I am sensible of the compliment paid me, and trust that in bringing before your notice these obervations on Pasteurellosis of the horse, I have not promulgated a subject which will prove uninteresting to you.

The Pasteurelloses are a group of diseases affecting not only all the domestic animals, but also the guinea-pig, the rabbit, the goat, the buffalo, wild animals, and birds, whose connecting link lies in the fact that their etiological factor in each case is an organism which possesses identical staining cultural and other characteristics. In this grouping are now included diseases, the relationship of which was not even suspected formerly. They are all types of hæmorrhagic Septicæmias and in spite of the numerous animal species affected the same processes are established in all, the same essential lesions are always recognised. There is a similarity in the mode of evolution of each disease and the clinical and anatomical manifestations are often superposable so that the termination of each may often be foretold.

It is to Lignières that we owe so much of our knowledge of this class of diseases and he has particularly worked up the affection in the horse.

The organisms of the group Pasteurella possess the following essential characteristics: Non-motile coccobacilli with clear centres and coloured poles, very polymorphic, non-sporulating, ærobic, do not stain by Gram's method, can be cultivated in meat bouillon on gelatine on gélose, but giving no visible cultivation on potato naturally acid, do not liquify gelatine nor coagulate milk, not making indol in pancreatic bouillon, not reddening the gelose of Würtz, giving in cultures an odour *sui generis* (Nocard and Leclainche).

The researches of Lignières have brought to light a very important property possessed by many of the Pasteurella organisms, and this constitutes a weighty point in looking at the diversity of the symptoms manifested. He has shown that several of this group of organisms can cause ephemeral affections of so slight a nature that the disease passes unperceived; but although no symptoms are manifested by the Pasteurella infection, yet their toxins reduce to *nil* the natural defences of the organism, so that they permit or rather facilitate a

secondary infection by organisms naturally present in the respiratory or alimentary tracts. These secondary infections may be multiple and varied. One can conceive then how these secondary infections complicate the study of the microbic invasions and what errors may arise.

The dog, in the disease called distemper, furnishes the best example of the pernicious influence of the Pasteurella organism. Distemper is due to a Pasteurella organism, but many of the clinical types of that disease are due to secondary infections. Lignières regularly, at will, brought on the pneumonic form of distemper by inoculating a dog with a cultivation of the Pasteurella and then exposing him to cold or immersing him in cold water and allowing him to shiver. This pneumonia is not caused by the Pasteurella but by an organism (the cocco-bacillus fœtidus ozenæ of Perez) normally present in the mouth and nasal cavities of the dog and not usually pathogenic.

The microbes both of the Pasteurella group and the secondary infections afford an illustration of the transition of organisms from saprophytes to parasites. They seem to be present in the soil and in the forage, then when they gain an entrance into an animal's system they acquire an exalted virulence, consequently we get many more cases infected from the first. After a time the virulence becomes diminished, the last cases of an outbreak are generally mild, and the disease is said to die out.

Pasteurella have been found in those three diseases which are often classed under the one name of influenza, viz., pink-eye, catarrhal fever, and equine contagious pleuro-pneumonia; and Lignières assert that "all the grave forms possess common characters and an air of parentage which permits of their being united into one group." Up till now they have been considered, as it were, independent units. "They are all general infections from the outset, but after a rapid increase the microbes tend to disappear from the blood and the parenchymata of the organs, in order to vegetate in some certain media only. The Pasteurella are difficult to find by direct examination, even although the evolution has been so rapid. In a number of animals they are no longer met with at the moment of death."

I have referred to secondary infections following upon the depressing action of the Pasteurella infection, and you can quite understand that in consequence of these multiple infections diverse clinical types are presented which from their dissimilarity have been thought to be different diseases and have received different names.

Under the one term influenza we have in this country included the three diseases—pink-eye, equine contagious pneumonia, and catarrhal fever, though they are generally looked upon as separate entities. There is a good deal of confusion about the term influenza, and attempts have been made to limit its scope. Dieckerhoff and Williams class the above three mentioned diseases under it. The German for pink-eye is pferdestanpe, for contagious pneumonia brustseuche, and for catarrhal fever scalma. Friedberger and Fröhner do not recognise scalma as a distinct disease, their influenza is only pink-eye, they look upon pneumonia as a separate disease. The French recognise three forms, viz., typhoid fever (pink-eye), infectious pneumonia, and grippe (catarrh) all under influenza.

For some time past I have looked upon these three diseases as presenting many features in common, and since Lignières researches were published I have endeavoured to test his conclusions by the light of practice.

I intend to give you some recent examples of the interchangability of these three forms, but first I give you an example which to my mind goes far towards proving his assertion that they are types of septicæmias, at least the pink-eye form is.

During the month of December, 1901, we had in the College Infirmary, amongst other patients, several affected with typical pink-eye. (It will be unnecessary for the purposes of this paper to detail the symptoms of that disease; I take it for granted that you know them). One of the patients not affected with pink-eye was a brown cart gelding which had been fired and blistered for lameness and had stood at the College in one box for six weeks. He was consequently under constant supervision. His box was quite near to some occupied by the pink-eye patients. During the course of one day (Dec. 11th) he was noticed to be lying down frequently, similar to a mild attack of colic. In the afternoon, noticing he had not eaten his midday feed, I examined him more closely. To my surprise I found his pulse to be 84 and weak, and the temperature 104·8; the respirations were a little accelerated, and the mucous membrane of the mouth was injected, and of a yellow brown colour. Next morning the pulse was 85 and the temperature 105·9, and the respirations 14, *i.e.*, not much above normal; the colour of the mucous membrane of the mouth was a deep yellowish-brown. The day following the pulse could not be felt at the jaw, but the heart was beating tumultously at the rate of 100 per minute, similar to what it does in all septic infections; the respirations were 19, shallow, and sighing; the mucous membrane of the mouth was brown and pasty-looking, that of the conjunctivæ pinkish and injected; there was no appetite, and the mouth exhaled a very fœtid odour. Since the preceding night a large fluctuating swelling the size of a football, but not quite so prominent, had appeared over the right shoulder; there was another swelling the size of a cocoanut behind the right elbow and still another one of about similar size on the back; the gluteal muscles were conspicuously flabby.

The animal died at 2 p.m. that day, practically about 48 hours from being noticed to be ill, and the post-mortem was made two hours afterwards. The subcutaneous tissue was in places infiltrated with an amber-coloured gelatinous serosity, particularly in the region of the above mentioned swellings. The stomach was about half full of a clear watery liquid containing some bits of masticated hay but no corn, one ascaris megalocephala found there, the external coats looked normal but the walls were thickened. The cuticular portion was normal, but the villous part was diffusely congested and of a bright strawberry colour, its mucous membrane easily tore off. The small intestines were normal, the cæcum was slightly congested, the first three portions of the colon were normal but the fourth part was inflamed, the lymphatic glands were swollen, soft, and hæmorrhagic. The liver and kidneys were congested, the spleen was normal. At the root of the lungs was a yellow gelatinous exudate extending along the œsophagus and aorta, and around the base of the heart. This gelatinous exudate was firmer than what one finds in anthrax. The lungs was congested with black blood, the trachea, large and small bronchi were all filled with a frothy spume. Subpleural hæmorrhages were present on that part of the lung just posterior to the heart on the left side; these hæmorrhages were chiefly about the size of a pea, but there were some diffuse ones. The heart was enlarged, the ventricles contained no blood, but each a small post-mortem clot, the valves were normal and there were no hæmorrhages on the endocardium. The blood was black but it coagulated slowly and brightened on exposure to air.

Briefly speaking, the post-mortem appearances suggested some resemblance to anthrax as it occurs in the horse, but I made sure by microscopic examination of the blood that it was not that disease. The gelatinous exudations and hæmorrhages would indicate a septicæ-

mia, and judging from the prevalence of pink eye at the time of the onset of the affection, strong evidence is I think adduced that this was a case of hæmorrhagic septicæmia due to infection from the pink eye patients. I could not trace the method of infection.

I remember one other case which presented very similar symptoms which came from a stud of 100 horses where contagious pneumonia is endemic, so to speak. He died in two or three days, and on post-mortem examination the lesions were even more suggestive of anthrax. No anthrax bacilli were found on microscopic examination of the blood, though the gelatinous exudate around the lymphatic glands along the attachment of the great colon, together with their hæmorrhagic condition were almost identical with what is found in anthrax in the horse.

I am comparing the appearances to those of anthrax because I think that disease is the *beau ideal* of a septicæmia. Within the past few weeks a case has been communicated to me of a horse which died after only four hours illness, and in which the post-mortem appearances would have served for a description of anthrax, the blood was oozing from the nostrils and anus etc., yet when I examined the blood no anthrax organisms were found but only a few cocco-bacilli. This case I venture to submit is also a case of hæmorrhagic septicæmia from infection with the Pasteurella organism.

I remember also one other similar case. We cannot be dogmatic from the above few cases, but I think we can go the length of saying that in the horse we meet with a septicæmia causing rapid death, not caused by the anthrax organism, possibly by a Pasteurella. Nocard and Leclainche give the duration of the peracute form as 24 to 36 hours.

Speaking of the interchangability of the three clinical form of Pasteurelloses of the horse, as a rule an epidemic assumes one of the three types and sticks to that, nevertheless one occasionally sees a coupling of the one with the other. Besides, one meets with what we may call abortive forms during an outbreak.

I have met with one or two outbreaks in which whilst one form of the above mentioned types predominated, one or two patients became affected with one of the other types. Some times ago a horse in a stable containing twenty-two horses became affected with pink eye. It rapidly spread, and all the animals with the exception of three, had more or less severe attacks of pink eye. Two of these three remained healthy, but one of them at the time the others were suffering from pink eye, instead of following the regular course, had a pretty bad attack of contagious pneumonia.

The contention may be urged that this was only a case of pneumonia complicating or supervening on pink-eye, but this case showed no eye symptoms, and the pneumonia was marked from the outset.

In another outbreak in a stud of twelve horses, last year a mare (Kitty) took contagious pneumonia (in which, however, pleurisy was the predominating lesion). She was removed to the College Infirmary to prevent the other horses catching it, and she remained there three weeks. Convalescence was slow, and when she returned home she had still her chest half full of water. She was placed in her old stall near to which was a grey gelding. On the seventh day after, the grey was noticed to be off her feed and on examination was found to have pneumonia. Four more horses became affected with typical contagious pneumonia, one other horse became feverish and off his food, he did not develop pneumonia but he suffered from exudative iritis and photophobia of both eyes. The pupils became obscured by a deposit of fibrinous lymph which after a time dropped down into the bottom of the anterior chamber. It then assumed a greenish-yellow colour and gradually disappeared. This resembled the condition known as periodic ophthalmia very much, and as if to lend colour to that statement the horse is reported to me as being now blind in one eye. Anyhow it is rather curious that if so his first attack should come on whilst contagious pneumonia was prevalent in the stable.

In a third outbreak pink eye broke out eight days after the introduction of a new horse. This new horse seemed healthy when purchased, with the exception of an enlarged submaxillary gland. After two or three days he developed an attack of epizootic catarrh or catarrhal fever, and two or three days after that two of the regular horses became infected with pink-eye. Shortly after that two more cases became similarly affected. We had no other case of pink-eye in our practice at the time, nor did I know of any other stable where it prevailed. This was in the same stable as the preceding outbreak, and the mare Kitty, the one which carried the infection back to the stable and caused the outbreak of contagious pneumonia, was one of those which took pink-eye. In her case, anyhow, an attack of contagious pneumonia one year did not render her immune to pink-eye the next.

In still another outbreak at a different stable where contagious pneumonia prevailed one mare showed all the classical symptoms of pink-eye and no pneumonia.

It has been my lot to have a good deal to do with contagious pneumonia. One of our clients has over a hundred horses stabled in one yard though in four separate stables. It is hardly an exaggeration to say that every new comer into the stable becomes affected with either epizootic catarrh or contagious pneumonia, one or the other. Many new horses are purchased, because Edinburgh is the distributing centre, so the opportunities for observation are not wanting. Moreover, many horses begin first of all by having an ordinary "cold," then they get a sore throat, and finish up by having pneumonia, which presents all the symptoms of the contagious type.

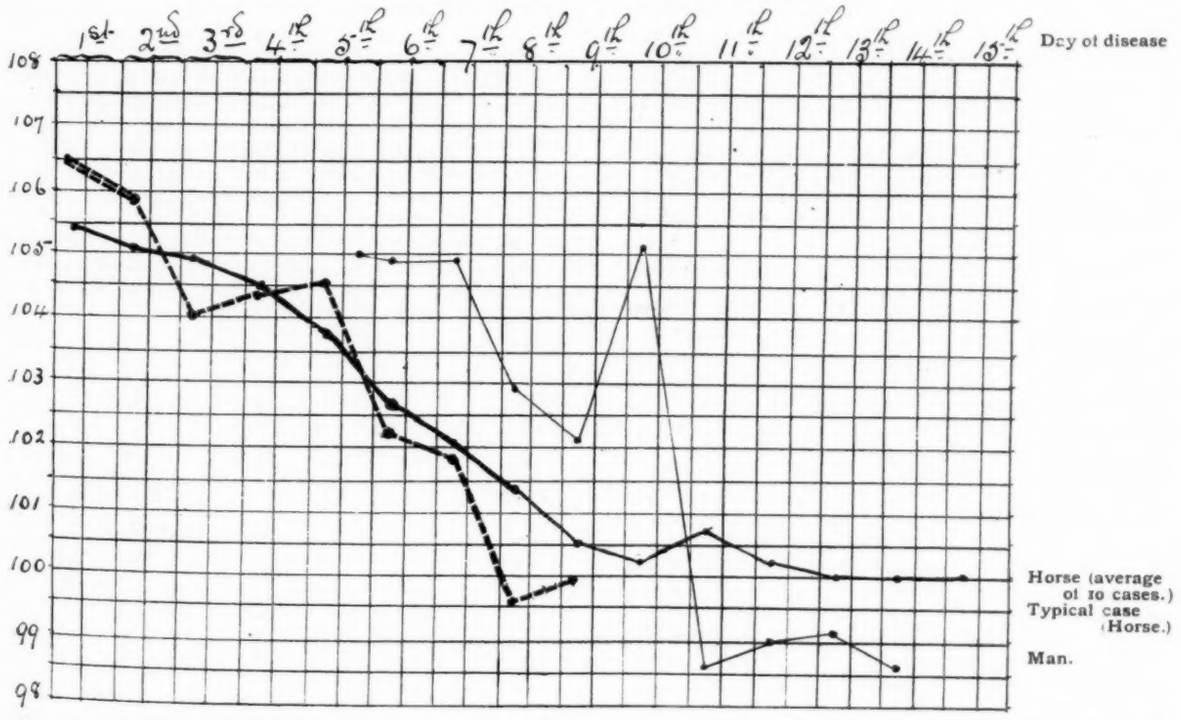
The above examples of various outbreaks lend colour I think to the statement that in the three diseases of the horse known as influenza, and comprising pink-eye, epizootic catarrh and pneumonia, there is something akin, but now we know the *rôle* of the Pasteurella organisms, and that of the secondary infections, the comprehension of the different clinical types is much simplified for us.

I am inclined to think that in the contagious pneumonia there is a plurality of organisms rather than the one (the diplococcus of Schütz) responsible for the lung lesions. I have prepared sections of lung and found them literally teeming with organisms, both those taking Gram's stain and those not doing so. Moreover, I have ceased to regard contagious pneumonia as an inflammation of the lungs pure and simple, and comparable to pneumonia of the human subject, for nearly all the fatal cases show well marked indication of gastro-enteritis in addition. I have known horses practically recovered from pneumonia suddenly become affected with enteritis and die in a short time. Others also have died quite suddenly from clots in the heart or large vessels.

Nocard and Leclainche class pneumonia as a complication of pink-eye, in fact they say it is the commonest one. Now I have seen pneumonia cases showing eye symptoms like pink-eye, but the commonest colour of the conjunctival mucous membrane is some shade of yellow, generally a dirty yellow. This I hold is evidence of a septicæmic affection, the old so-called hæmatogenous jaundice and not due to catarrh of the bile duct. Another thing which strengthens me in that belief is that for the first few days at any rate, one can hardly ever demonstrate any pneumonic lesions either by percussion or auscultation. This conclusion I have arrived at from numbers of observations. The lung chiefly affected is, I think the left, at that part just behind the heart. A

word about the temperature curve in pneumonia ; in the horse the thermal curve is quite different to that found in a case of pneumonia in man. In human pneumonia the temperature rises rapidly, and having reached the fastigium remains constant, then about the seventh or ninth day there is a sudden drop down to nearly normal. The crisis is the most remarkable single phenomenon of human pneumonia. In equine pneumonia the onset is perhaps as sudden, the temperature very quickly rising to 105° or 106°, but in an ordinary case there is no period of crisis; the temperature as a rule falls a little each day, though in some cases about the fifth or sixth day there is a drop of 1·5 to 2 degrees. I hand round a chart with the thermal curve in a typical pneumonia case.

Temperature Chart of Pneumonia in Horses and Man.



In contagious pneumonia we meet with varying degrees of the severity of the affection. The discharge from the nose I consider a good guide to the intensity of the lesion. Mild cases show practically no discharge, cases a little more severe have an amber coloured discharge, not well seen if a nasal catarrh complicates matters. More severe cases still have a discharge which is well defined by the word prune juice like. Many of you have no doubt seen cases answering to the above degrees of severity. There is still however, I think, another more severe condition, perhaps some of you have met with it also. I have come across some dozen or so of cases where the discharge was actual blood. The course of such cases was somewhat as follows: The animal apparently well one day was noticed to be blowing heavily the next, and on examining his nostrils black blood is found running down in more or less quantity. The pulse is about 80 or 90 and very weak, the temperature 105° or so. The symptoms became quickly aggravated and the animal generally died in about 24 or 48 hours. Most of the cases came from badly ventilated stables. Few cases rallied, but one or two did only to be affected later on with gangrene of the lung. This of course caused death.

On post-mortem the lungs are black and hepatised. Microscopic sections of these lungs show rather a curious lesion. Under a less power one sees bundles of acricular crystals which shoot out in all directions. These are the most striking feature and arrest the eye at once. The pneumonic changes proper are not very marked, the vessels are congested, and here and there we find a few catarrhal cells or leucocytes *plus* fibrin in the alveoli.

This peculiar lesion is not described in any of the text books, either English or Continental, with which I am acquainted, nor can I find in any works on pathology any reference to it. I have shown the sections to several pathologists, all of whom on first sight thought the appearance was due to something extraneous in the preparation. Two gentlemen (one of whom was Dr. Dean, examiner in Pathology to the R.C.V.S.) to whom I gave pieces of lung to cut and stain for themselves, have obtained similar results. Dr. Dean says it is something he has not met with before and it is "probably not an artefact." I had come across the lesion so constantly in urine that I felt sure it was not in the cutting or mounting of them. What are the crystals? That is a difficult question to answer.

I have examined them by the polariscope, and they are not inorganic. The most feasible explanation I can suggest is that they are some derivative of the hæmoglobin of the blood, and that they are produced at death. I am convinced in my own mind that these cases showing the hæmorrhage from the nostrils are cases of contagious pneumonia of an extremely virulent type, and I think it perhaps likely that the toxins of the organism have caused solution of the red cells and liberation of the pigment into the plasma. You will see under the microscope specimens showing the crystals, and also in the microphotograph. The pigment in the plasma will most likely have crystalised at death. On examination of the sections with the spectroscope to see if the crystals were hæmoglobin, nothing could be made out. They are too thin and the stain masks the effect.

With regard to the actual cause of contagious pneumonia our views must be modified since Lignières' researches. The diplococcus of Schütz, which was given

as the organism responsible for the pneumonia (and which, by the way, has been shown to be identical with the streptococcus of strangles) intervenes only secondarily. The Pasteurella prepares the lung tissue, and the diplococcus of Schütz which is present without causing any lesions in the healthy lung, becomes pathogenic. It invades the lung and produces its characteristic effects. It is a secondary infection then, on top of this secondary infection we sometimes have a tertiary one in the shape of gangrene.

A rather curious fact in connection with the different clinical manifestations of equine Pasteurelloses is the difference in the mortality. The catarrhal form is quite a benign form, and I cannot recollect a death from this if uncomplicated. The mortality from pink-eye varies; some years the attacks are mild and hardly a death is recorded; at other times, especially amongst cart horses, the mortality is pretty heavy. The mortality from pneumonia is, I think, greater than the other two forms, but this is to a very large extent dependant on the sanitary surroundings; if good hygienic principles are not observed, gangrene of the lungs followed by certain death is the result.

A curious feature about these three forms is the difference in the degree of contagiosity; pink-eye at times ravages a whole county, pneumonia is generally endemic, epizootic catarrh remains often confined to one stable.

As sequelæ to contagious pneumonia, in addition to the more common ones, I have met with one case each of broken wind, alopecia, and fibrinous iritis. In the case of alopecia the animal after an attack was left extremely weak; in a few days the hair on various parts of the body began to fall out, the patches were of irregular size and shape, many were completeiy denuded of hair, others were only partially so. There was practically no itching, and the skin in the majority of the spots was as smooth as if it had been shaved; the insides of the legs and the neck were almost bare and quite smooth. After a time a new growth of normally coloured hair re-covered the spots.

The age of animals affected with these three clinical types is worth noticing. Pink-eye affects animals of all ages and seemingly indiscriminately, pneumonia has a preference for the younger animals. I am sorry I cannot give you the exact figures of the proportion of animals attacked at the different ages, but speaking from memory I should say that more than half the cases are five or six years old. This fact is interesting, we know that as a horse gets older he becomes more immune to strangles; similarly, then, he seems to become less liable to be attacked with the same organism causing pneumonia. I cannot say if an attack of strangles protects a horse against pneumonia, but I look upon one attack of pneumonia as conferring a good deal of immunity. Cadiot relates an interesting incident regarding immunity. In a French regiment of 780 horses 64 became affected with contagious pneumonia, mostly four and six year olds; very curiously, out of 79 five year olds not one became affected. It turned out that these 79 horses had the year before at the Remount depôt been concerned in an outbreak of contagious pneumonia and that many of them had had it.

Let us just glance at the methods of infection in the three forms. One is apt to refer an outbreak always to aerial infection, and certainly this is one method of spread. A horse, say, from a dealer's stable or a livery stable if brought into another stable rapidly infects the horses therein, the spread of the disease indicating the contagium to be volatile. I think, however, that we are a little apt to lose sight of another mode of entry of the organism into the system, viz., by the digestive tract. It seems to me very likely that those outbreaks which occur say on farms away from contact with other horses, are thus explainable. Once the organism has gained entrance to an animal its virulence may became increased. These saprophytic organisms vegetate in the soil, so it seems natural to conclude that the fodder becomes contaminated. The food stuffs or water may become infected also by the dust from the intestinal discharges, or from the nasal discharge of affected animals. The sojourn of an animal for a short time in an infected stable, loose box, or railway truck will give the disease, but here the demonstration of the mode of infection is not always easy. Another very curious method of infection is by coition, the male animal in these cases presenting no signs of illness. This only takes place in pink-eye as far as I am aware.

There is a disease described by Dieckerhoff under the title of Ephemeral Pneumonia which is characterised by sudden rise of temperature, quickened pulse, and great increase in the number of respiratons. On auscultation nothing much can be made out except a roughness in the vesicular murmur. In about 24 hours all the symptoms have disappeared. Nocard and Leclainche also propose to class this as a Pasteurella infection. As to its etiology I prefer to keep an open mind. The disease is not exactly like what in this country is called simple fever, because the great increase in the number of respirations is the most striking point. The following case is a good example. A black cart horse standing in the stable all day, towards evening began to "blow." I saw him at 7·30 p.m. and his temperature then was 106·9 his pulse 80, and his respirations 55 per minute. Next morning at 10 a.m. his temperature was 101·2, his pulse 42, and respirations only 14. Truly a remarkable drop all round.

A good discussion followed, the following members taking part: Messrs. Hunter, Davidson, Dudgeon, Gofton, Aitken, Mitchell, and Craik.

Professor TAYLOR having replied, he was accorded a hearty vote of thanks for his interesting paper, a vote of thanks to the retiring President concluded the meeting.

E. R. GIBSON. *Hon. Sec.*

CANCER AND ITS ORIGIN.

The Bradshaw Lecture delivered before the Royal College of Surgeons of England on Dec. 9th, 1903.

By HENRY MORRIS, M.A., F.R.C.S., Eng.

[ABSTRACT].

(Continued from p. 394).

I propose now to examine, as well as the time will permit, the evidence in favour of the separate parts of "the tumour germ theory"—namely, (1) the existence of congenital matrices of embryonic cells; (2) the existence of matrices of post-natal formation, (3) the continuance of these matrices for a time in a dormant state; and (4) the causes which arouse the matrices into activity.

1. What proof do we possess of the existence of congenital matrices of embryonic cells? It is hardly necessary to enumerate the many instances of "vestiges" and "fœtal rests" which are found in the human body, as they now form a brief chapter in the latest text-books on anatomy. Tracts of embryonic epithelium are well-known to occur in the tongue, gums, face, scalp, neck, brain, eye, testicle, broad ligament, ovary, and along the lines of coalescence of the trunk.

Many matrices are derived, we know, from portions of embryonic skin becoming buried in the mesoblast in the process of closure. According to Ebstein it is not uncommon to find in newly born infants isolated pearls of epithelial cells buried in the connective tissue by inclusion. Hexhl looked upon subcutaneous atheroma as the product of tissue-proliferation from such an island of

epithelial tissue or portion of epidermis with perhaps a single hair. Rests of connective tissue occur as islets of cartilage in the neighbourhood of the epiphysial lines of long bones, especially in rickety subjects. Matrices of embryonic tissue may be met with in normal situations for such tissue or in abnormal; when in abnormal the matrix is said to be "misplaced." Bodies identical in structure with the suprarenal gland are found not only in connection with the capsule, the cortex and the hilum of the kidney, and in the perinephric tissue, but also beneath the capsule of the liver, in the broad ligaments of a fœtal uterus, in the inguinal canal, and in some fatty tumours of the spermatic cord. That they have not more frequently been met with than is the case is probably due to the fact that they have not been looked for, and Bayard Holmes states that remnants of the adrenal body can be discovered in various parts of the genito-urinary tract in 90 per cent. of all post-mortem examinations.

Dr. John Beard in a lecture on the Embryological Aspects and Etiology of Cancer states that in elasmobranch fishes (and he implies that the same conditions exist in man) a considerable percentage of the primary germ cells fail to reach their normal position, the germinal ridge; that many of these vagrant germ cells degenerate and disappear, but all do not; and that the common situations for the origin of cancer are the very ones in which vagrant germ cells are usually met with.

2. The occurrence of matrices of embryonic tissue of post-natal origin is capable of completely satisfactory explanation. Leo Loeb has described how in the healing of a superficial wound the epithelium of the skin on either side of the wound was seen to have fused itself into a cell mass which sent off branches not only across the wound but down into the blood clot and the connective tissue, especially if the connective tissue had been injured, and these branches of the cell mass even burrowed through cartilage. It is intelligible enough, therefore, how in the cicatrisation of ulcers and wounds generally some of the epithelial buds become "isolated," and being thus shut off from their proper anatomical relations and from their normal function may, under suitable stimulation, develop into carcinoma. Epithelial cells not utilised in the process of epidermisation and becoming buried in the scar tissue may when brought under the influence of the needful exciting cause give rise to carcinoma. This sequestration of unused cells may occur during healing after burns, chronic ulcers, lupoid and syphilitic sores, fissures, sinuses, fistulæ, compound fractures, cracked lips, the excoriations from soot, tar, or paraffin, and after lacerations and ulcerations of the cervix uteri; also after the alternate breaking down and healing in such dlseases as ichthyosis of the tongue, cheek, vulva, vagina, and other ulcers caused by carious teeth, as well as after wounds, whether operation wounds or otherwise.

In like manner a matrix may be produced from which sarcoma may arise. In parts which have been the seat of chronic inflammation and in scars, unspecialised connective tissue cells may be buried, and will be there ready to grow into a sarcoma under the influence of a sufficient exciting cause. Some of the embryonic cells during the process of repair of a fracture may fail to attain to the perfection of their parent cells and will thus remain in the part in a latent condition, like the congenital matrix of embryonic cells predicated by the original theory of Cohnheim.

3. What proof have we that a matrix of embryonic tissue, whether of congenital or post-natal origin, can remain for a length of time in a quiescent state and then suddenly start into active growth under the influence of some stimulus? This is actually witnessed in the growth and development of the teeth, the bones, the skin and its appendages, the larynx, the mammæ, and the organs of generation, the cells of which remain for years in a state of incomplete differentiation and then normally, under the physiological stimulus which occurs at puberty, take on rapid proliferation. Pathologically the same suddenly aroused activity is seen in certain epithelial tumours which are rarely noticed before puberty, such as dermoids, branchial cysts, and mammary adenomata. Rudimentary organs and accessory organs which hitherto as to growth had been stationary as well as functionless become the starting points of tumours, more especially at and after puberty.

4. The causes which arouse a matrix into activity are numerous. For a matrix of embryonic cells to be transformed into a tumour it must be aroused into activity by some exciting agent. We must now inquire what these exciting agents are and in what relation the commonly ascribed causes of cancer stand to the "matrix" or "tumour germ" theory. The stimuli which arouse the cells of the dormant matrix to active proliferation are either general or local, congenital or acquired, and the chief amongst them are the chief amongst them are the alleged common causes of cancer. Let us consider four of them in some detail—namely, heredity, age, traumatism, and chronic irritation.

(*a*) *Heredity.*—A case like that of Friedreich, where a woman with carcinoma gave birth to a child affected with carcinoma, is altogether exceptional.

Statistics hitherto have given insufficient and inaccurate information concerning the frequency with which the offspring of parents carcinomatous at the moment of conception or delivery have been affected with carcinoma. There is, however, no more reason to doubt that an apitude is transmitted from parent to child for the development of carcinoma and sarcoma than for denying an hereditary disposition to various other diseases. This aptitude, or predisposition to malignant disease, has been appropriately described as "a diminution of the physiological resistance of the tissues" which permits of their being invaded and destroyed by the proliferating cells of the tumour matrix. In the great majority of cases (in Cohnheim's view in all cases) the matrix itself is of congenital origin, but though congenital, it may never be excited into activity. In others, the matrix is of post-natal origin, and if the necessary stimulus arises and the tissue resistance is weak cancer will occur, even in persons in whose family cancer had hitherto been absent.

In a sense, then, all tumours are congenital or hereditary, except such as are derived from a post-natal matrix, and even in these cases the matrix will not develop unless the normal physiological resistance of the tissues is weakened by some cause, which may be hereditary, but which, on the contrary, may not be so. For a tumour to be inherited there must be a congenital matrix of embryonic cells, or there must be transmitted from parent to offspring a lack of physiological resistance either of the whole of the tissues of the body, or at least of those adjacent to the matrix, whether the matrix be congenital or acquired. The exciting cause will in most cases be acquired, not inherited. If it be granted that there is a family tendency to vestiges, rests, or matrices of embryonic cells it is at once intelligible how such an hereditariness may lead to mammary carcinoma in one member, uterine carcinoma in another, squamous-celled carcinoma in another, and sarcoma in a fourth member of the same family.

(*b*) *Age.*—Carcinoma is admittedly a disease of middle-aged and elderly persons, and of senile tissues or organs like the uterus and breast after the child-bearing age. But on the "tumour germ" theory there is no difficulty in explaining the occasional occurrence of carcinoma in very early life, or at any period of life if the physiological resistance is especially weak in the individual and the matrix irritant or exciting cause is very

considerable. Sarcoma occurs in much younger life than carcinoma and is most frequent in children and young adults, but no age is exempt from it

There may be in the same person several congenital matrices, one or more of which may be of the epithelial type, one or more of the connective-tissue kind. During childhood and young adult life the exciting cause, or matrix stimulus, will start into activity a connective-tissue "tumour germ," but during mature life and in advanced age the same stimulus will arouse by preference a matrix of embryonic epithelial cells, because the epithelium is more active and vigorous than the connective tissue at the later periods of life.

Assuming the presence of a matrix of each kind in the same individual and that middle life has been reached without the occurrence of any matrix-arousing stimulus, but that after this period some *general* exciting cause comes into existence, the matrix of epithelial cells will most likely be the one to respond and a carcinoma, not a sarcoma, will result ; but there may be the simultaneous appearance of different kinds of tumours. Should the stimulus be a *local*, not a general one, the matrix upon which alone it acts will be aroused and a carcinoma or a sarcoma will be developed according to the type of cells forming the matrix which is stimulated.

With advancing age, or senility of tissue, come impaired vitality and lessened power to resist the effects of injury, hence the diminution of the physiological resistance of the tissues and the increased proneness to the formation of cancer. Little is known, it must be admitted, of what this "physiological resistance" really consists; what this power is which holds in check altogether, or for a long time, the proliferating tendency or force of a tumour matrix, as it does for years the growth and development of the mammary glands, the organs of generation, the larynx, the teeth, and the beard. The existence of such a force was, however, demonstrated by Cohnheim and Maas who introduced pieces of young periosteum into the jugular veins of animals. The periosteum produced bone which was always limited in size to the lumen of the vessel in which the periosteum embolus became impacted ; the bone never extended beyond the vessel walls, a physiological resistance restricted it, no further growth ensued, and in time the ossified periosteal graft was absorbed. The same investigators showed that transplanted grafts of embryonal tissue succeed, whereas grafts of mature tissues are invariably removed by absorption in a short time. Embryonic tissue—fœtal cartilage, for example—taken from animals before they are born and engrafted into rabbits proliferated abundantly ; grafts from benign tumours only grew to a very limited extent, if at all, and were then removed by absorption. Grafts of malignant tumours have almost, without exception, failed, though composed as they are of embryonic tissues. This seems to show that the physiological resistance to malignant cell proliferation is, happily, more effective than against fœtal embryonic tissues or against the embryonic tissue of benign tumours. The weakness or want of this physiological resistance, besides being sometimes inherited and often a consequence of age, can be acquired through injury, irritation, inflammation, and other pathological, general, or local causes.

(c) Traumatism is often said to cause malignant disease, just as it used to be said to cause inflammation and suppuration. But just as no amount of injury will of itself excite suppuration without the presence of pathogenic and pyogenic organisms in the injured tissues, so no amount of injury will give rise to a tumour without the presence of a matrix of embryonic tissue of either congenital or post-natal origin. No so-called exciting cause will produce cancer or sarcoma in the absence of the "tumour germ" of epithelial cells or connective-tissue cells respectively. But given the presence of such a matrix then injury of any kind, irritation of any kind —mechanical, chemical, thermal or microbic—may excite cancer formation both by arousing the matrix into activity and by diminishing the physiological resistance of the surrounding or adjacent tissues. Whether the cancer follows the injury quickly or tardily depends upon the intensity of its effects upon the matrix, or upon the degree to which the physiological resistance or, what is probably the same thing, the nutrition is affected, or upon both these conditions.

(d) Prolonged irritation and inflammation may (1) excite a matrix of congenital tissue to proliferate ; (2) may diminish physiological resistance of the tissues around it; or (3) may provide a post-natal matrix of embryonic epithelial or connective tissue cells and thus originate the essential factor of malignant disease as well as stimulate it into proliferative activity. Carcinoma of the breast following eczema of the nipple is an instance of malignant disease aroused by chronic irritation and inflammation. Epithelioma of the tongue following long-standing ichthyosis of that organ is another. They are typical illustrations of agents which act in each of the three ways just mentioned. Yet that neither of these pathological irritants is of itself the true and essential cause of carcinoma is proved by cases in my own practice. In 1880 I published two of the earliest reported cases of chronic eczema of the nipple (Paget's disease). Out of 305 cases of mammary diseases which up to that time I had seen in my hospital cancer outpatient practice, eczema of the nipple was met with in eight cases, in six of which there was no cancer present in the mammæ. In only two cases then out of eight did carcinoma of the breast follow the chronic eczema of the nipple and areola. It is certain, therefore, that something more than the long-standing irritation of chronic eczema is needful for the development of mammary cancer, and this sometimes is a "tumour matrix" in the breast. If due to irritation alone, why did not carcinoma occur in all the eight cases equally ?

If during a stage of cicatrisation of part of the eczematous area a local post-natal matrix had formed, the carcinoma, beginning as it might then have done in the skin, would have been a true squamous-celled epithelioma, not a glandular carcinoma. In one of these two cases there was a wart in close proximity to the eczematous areola, but though a wart is apt in certain circumstances to become an epithelioma, it was the breast, not the wart, we must assume, which contained the tumour matrix most readily provoked into riotous epithelial proliferation. It can hardly be maintained that the carcinoma was due to prolonged Irritation of the eczema upon a breast in which the senility of the organ had very specially diminished physiological resistance (or the "tissue tension") of the part, because one of the women referred to was hardly 30 years old and the other was only just 40 years of age.

If it be granted that for cancer to form a matrix must exist, and that without such a matrix no irritation, however prolonged, can cause the formation of a malignant new growth, we can at once explain how it is that a carcinoma does sometimes originate near a lupoid or syphilitic sore, not in it, near an ichthyotic patch, not in it, near an ulcerated nipple and areola, not in them. A tumour matrix within the limits of an inflammation receives an increased blood-supply and this of itself is an incitement to active cell proliferation. Friedländer has shown by experiments that inflammation diminishes physiological resistance; so that persons born with, or acquiring after birth, a tumour matrix are more likely to become the subjects of malignant disease if the adjacent structures become the seat of inflammation or irritation; whereas without such a pathological irritant and the consequent loss of power of resistance of the neighbouring tissues the tumour matrix might remain latent

throughout life. Carcinoma undoubtedly attacks parts and organs that are frequently the seat of prolonged and repeated irritation.

I is unnecessary to dwell on the several other alleged causative factors of cancer. Sex, race, climate, soil, food, locality, mental worry, and so on can under the "tumour germ" theory be nothing more than secondary or subordinate agents, acting either by way of stimulating a pre-existing matrix or by diminishing the physiological resistance of the structures, or both. To what extent they have the power of doing either we do not know, and we must await the compilation of more accurate statistics than have hitherto been made.

5. The validity of the premisses of the "tumour germ" or "matrix" theory being assured, let us see whether the conclusion to which the theory leads can be established by actual facts. This conclusion is that cancerous as well as benign tumours spring from "inclusion masses"—some of which are "displaced" masses—of embryonic cells either of congenital or post-natal origin. A large variety of benign tumours which are well known to arise from congenital matrices might be quoted.

(*a*) We now know that accessory spleens, accessory ovaries, accessory thyroids, accessory suprarenals, accessory mammæ and outlying portions of the mammary gland, and remains of the Wolffian body are all responsible for tumour formations. All that is known of teratomata—endogenous and exogenous—lends support to the Cohnheim theory. All endogenous teratomata or teratoid tumours, including branchial cysts, and the simplest as well as the most complex forms of dermoids, undoubtedly arise from congenital embryonic matrices. Skin dermoids are the most frequent form of fœtal inclusions and consist of portions of embryonal skin isolated and buried in mesoblastic tissues; dermoids which contain mucous membrane are derived from a matrix of hypoblastic origin; whilst dermoids with complicated contents, such as bone, cartilage, and portions of organs, can originate only from a displaced matrix representing different tissues and organs. Or, conversely stated, matrices of epiblastic, hypoblastic, and of complex origin misplaced into abnormal situations are known to give rise respectively to dermoids containing sebaceous material and hair, to mucus, and to complex dermoids like those of the testis, the ovary, and other organs. Branchial cysts are derived from the remnants of branchial clefts and these remnants, like the "inclusion" matrices of dermoids, may remain dormant for an indefinite time or become the seat of tissue growth through the increased physiological function which commences at puberty or at any subsequent time on being stimulated by some extrinsic exciting cause. Rests of the kidney itself and rests of the suprarenal capsule are now known to be the starting points of certain benign and malignant renal tumours; and similar rests, and also remains of the Wolffian body, are the source of origin of certain paranephric tumours. Grawitz traced tumours of the kidney to islets of separated and displaced renal tissue. Outlying portions of the mammary gland are the possible source of those adenomata which are occasionally found at the margin of the breast. If remnants of a glandular structure resembling the mamma are recognised, as they now are, as occurring outside and also within the limits of the organ, is it not most probable that similar isolated or sequestrated portions occur within the capsule of the other glands and will be met with when they come to be more carefully sought for? Indeed, the view is at present held that such sequestrated and encapsuled portions of the glands are the source of fibro-adenomata, adenomata and small cystic tumours found in he liver, parotid, kidney, and other organs, as well as in the mammary glands.

(*b*) There is no lack of evidence, either, that tumours are formed from matrices of post-natal origin, We know that tumour germs arise as the outcome of surface epithelium, hair follicles, or portions of skin forced into the deeper tissues by punctured and other wounds. From such sequestrated fragments of tissue "implantation cysts" are formed—a group of small tumours allied in character to the sequestration dermoids derived from matrices of congenital origin. These "implantation cysts" when occurring on the fingers are known as "digital dermoids." Others following lacerated wounds have been met with on the scalp containing hair and sebaceous material. Cysts of the iris following mechanical injury have been found to contain sebaceous matter and in one recorded case an eyelash. Cysts lined wifh layers of cells like those on the anterior surface of the conjunctiva have been known to form in the cornea after gunshot and other accidental wounds, as well as after operations for cataract, and are almost certainly derived from conjunctival epithelium forced into the deeper layers of the cornea.

(*c*) It has been unquestionably proved that the "tumour germ" theory correctly accounts for the origin of many varieties of benign solid tumours and of dermoids and other cystic tumours, and we must now try to answer the question with which we are especially concerned: Does it not afford the true explanation of the etiology of malignant tumours also? In attempting to reply to this question it may first be pointed out that regions and organs in which embryonal inclusions are specially likely to occur are also common situations of primary malignant diseases. To realise the truth of this statement we have but to recall the frequency of sarcoma near the ends of long bones, the occurrence of myeloid and periosteal sarcoma and epithelioma in the mandible and upper jaw, of carcinoma of the uterus, œsophagus, stomach, and rectum, and tongue and mouth. In the lip the commonest seat of origin of carcinoma is along the line of junction of the skin and mucous membrane. In the rectum the parts at or near the junction of the skin and mucous membrane, and those near the union of the proctodæum (cloaca) and the hind gut, are the areas of the greatest liability to carcinoma. In the stomach the pylorus and its immediate region are the chief seats of carcinoma, and it is here, as Cohnheim has pointed out, that the anatomical structure of the organ varies most; it is here also that there is the greatest liability to pathological changes and thus to the development of post-natal as well as of congenital matrices. Such anatomical areas as those just mentioned, like the cervix uteri, are zones of transitional epithelium and are provided with glands not always of normal functional perfection; in these so-called "cancer belts" we may expectantly look for 'tumour germs" in the form of isolated congenital nests of embryonic cells.

Primary carcinoma originating in a situation where no epithelium normally exists sometimes springs from a previously undetected dermoid cyst or dermoid-cyst matrix. A displaced matrix of epithelial cells isolated from its own blastodermic tissue and buried in the mesoblast, if it becomes a tumour, will develop into one of an epithelial character. Tumour cells, like normal cells, always breed true. Connective-tissue cells cannot produce epithelial cells, and epithelial cells invariably produce their own kind and no other. Virchow when he met with a primary carcinoma in the tibia, not recognising this physiological law, believed the cells of the carcinoma were produced from the connective tissue at the spot where he found them and called them epithelioid. The knowledge of this law as applied to a carcinoma developed from a displaced or misplaced epiblastic matrix which has been roused into proliferative activity by some exciting agent corrects such a histological error

and explains the occurrence of the heteroplastic tumour It also explains how an "endothelioma," which resembles carcinoma in its characters, is developed as a primary new growth in serous membranes and lymphatic glands —structures which are of mesoblastic origin and in which, therefore, no epithelium normally exists; it likewise explains how a glioma, which in its anatomical and clinical characters is a sarcoma, can occur in the brain or retina which are derived from the epiblast.

6. We must next inquire whether the "tumour germ" theory can account for the transformation of a benign into a malignant new growth. We need not stop to inquire what evidence there is that this transformation ever takes place. The fact of its doing so is no longer, as formerly it was, disputed. Warts, moles, dermoids, fibromata of the skin, uterine myomata, and papillomata in various situations might be quoted in proof of benign tumours passing into malignant.

The change to malignancy may originate in a matrix of tissue derived from the organ or part affected and which has become sequestrated and inclosed within the structure of the benign tumour during the process of the growth of the tumour. Or in the process of its growth some of the daughter cells within the benign tumour itself may fail to attain the degree of differentiation of the parent cells and may subsequently develop on their own account into a malignant neoplasm. A woman under my care some years ago was the subject of an intramural uterine fibro myoma which became the seat of colloid carcinoma. The case was published by Mr. W. Roger Williams who mentions that a few cases of carcinoma originating in uterine fibroids had been recorded by Virchow and others. It is probable that in my case the carcinoma, which was clearly glandular, was developed from the deep extremity of one of the utricular glands sequestrated in the process of the growth of the fibro-myoma and which thus came to form an epithelial tumour germ—just as glandular elements have been observed in the analogous new growths of the prostate.

Other Evidence in Favour of the "Tumour Germ" Theory.

The matrix theory enables us to understand the long intervals which occur after removal of cancer by operation before recurrence takes place, for just as the "tumour germ" of the primary growth remained for years unaroused, so a single cell or a few cells migrated to a lymph gland or duct or into the areolar tissue some distance away may continue quiescent owing to the efficiency of the physiological resistance of the surrounding tissue. The cell or cells are the "tumour germ" of the recurrent growth as the original matrix was of the primary, and are subject to the same kind of restraint and respond to the same kind of stimuli.

The "tumour germ" theory also explains the so-called recurrences, after either short or very long intervals, in parts or organs away from the original site and out of the line of direct lymphatic infection. It explains, too, the development in the same percon of two malignant tumours of different blastodermic sources and of their existing simultaneously, or of one occuring after the other has been excised.

Lastly, the "tumour germ" theory affords a better explanation of the local extension of carcinoma than any other which has been put forward. It ascribes it to cell proliferation of the margins of the matrix; the growth of the tumour to the multiplication of the matrix cells, not to the "reversion" or conversion of the pre-existing mature epithelium into tumour tissue.

The foregoing are some of the reasons for regarding the "tumour germ" theory as the most, indeed, in my opinion as the only, probably correct theory of cancer origin which has ever been put forward—as the only hypothesis which is consistent with all the known facts of cancer. Hitherto, what would seem the strongest evidence against the "tumour germ" theory is that which seems the strongest evidence in favour of the microbic theory—namely the successful serial experiments upon mice with cancer tumour tissue which were carried out by Morau in 1893 and have been recently repeated by Jensen and Borrel. But Borrel admits that his successful series was only obtained by treating at each "*passage*" a number of mice, varying from 12 to 15. This is of much importance, because many mice were found to be unaffected. The proportion of successes to the total number of animals experimented upon at each "*passage*" in the series ought to be given by each experimenter. The tendency to the development of cancer in mice would seem to be strong, judging from what is stated in Borrel's paper as to the frequency of cancer in certain caged mice not operated upon.

Up to the present time the microbic theory has not advanced our knowledge of the cause of cancer one iota in spite of all the talent and skill which have been employed upon it, whereas every year facts in support of the "tumour germ theory" are discovered almost without being sought for. Excepting for these aforesaid recently discovered facts our knowledge of the causation and nature of cancer would be no greater now than it was 30 years ago; indeed, the very facts about cancer which were recognised and established a generation ago are to-day the strongest *a priori* arguments against the microbic origin of cancerous diseases. The truth is that for the last 10 or 15 years cancer research has been too exclusively directed to microbes and too little to cancer. The attention given to cancer research by many able workers has been too restricted to the search for the problematical "cancer organism" to allow of much thought being directed to the other factors in cancer. But what is wanted, also, is that some equally skilled research work should be directed to other questions—embryological, morphological, chemical and functional—to such subjects, for example, as those to which Marshall drew attention in his suggestive Morton lecture, namely, the chemistry of cancer tissue, the structure and changes in the stroma, the minute anatomical relation of nerves to the cancer cells, and of the effect of the loss of control of the nerves over them.

Future researches may reveal a method whereby a special inhibitory influence may be brought to bear upon the cells of the "tumour matrix" on the lines tried by Foulerton, or a restraining, even a strangulating, effect upon the tumour cells by the tumoar matrix may be produced, as suggested by Marshall. But in any case we can reasonably hope and expect that the organised researches now commenced by the Royal Colleges will in time yield much information as to how to prevent cancer by teaching us what are the real agencies which stimulate it into existence.

Kicking—No evidence of vice

The dog's first bite is a well-known institution, much more gratifying to the animal and its owner than to the person on whom it exercises its privilege. But apparently there is also a horse's first kick to be allowed for. Recently in Newcastle a cycling case was heard in which a clubman sued for damages for injuries received from the kick of a horse. The rider met a horse which shied across the road and kicked him, inflicting a good deal of damage on the wheelman and his machine. The defence offered was that the horse is a domestic animal, and consequently the owner must be shown to have known of its vicious nature before he could be made responsible for its misconduct. As no proof of the owner's knowledge of the animal's propensities was adduced, the judge, agreeing with the argument of the defence, dismissed the case.

DISEASES OF ANIMALS ACTS 1894 TO 1903.

SUMMARY OF RETURNS, WEEK ENDING DECEMBER 26, 1903.

Period.	Anthrax.		Foot-and-Mouth Disease.		Glanders (including Farcy)		Rabies. Cases Confirmed.		Swine Fever.	
	Out-breaks.	Animals.	Out-breaks	Animals.	Out-breaks	Animals.	Dogs.	Other	Out-breaks.	Slaughtered *
Week ended December 26, 1903 ..	15	16	..	..	24	37	..	..	21	105
Corresponding week in 1902 ..	12	16	..	..	17	27	..	..	27	186
Corresponding week in 1901 ..	18	33	..	..	17	54	..	..	27	126
Corresponding week in 1900 ..	15	31	..	..	13	21	..	..	36	425
Total for 52 weeks, 1903	761	1127	..	..	1463	2490	..	..	1478	7933
Corresponding period in 1902 ..	678	1032	1	120	1155	2040	13	12	1688	8263
Corresponding period in 1901 ..	651	971	12	669	1347	2370	1	1	3140	15237
Corresponding period in 1900 ..	571	956	21	266	1119	1858	6	5	1940	17933

NOTE.—The figures for the Current Year are approximate only. * As Diseased or Exposed to Infection.
Board of Agriculture and Fisheries, Dec. 29th, 1903.

Personal.

We regret to hear that Lieut.-Colonel J. A. Nunn broke his collar-bone while hunting with the Whaddon Chase Hounds on December 26th.

JARVIE-MUNRO.—At St. Andrew's Church, Lamesley, Dec. 30th, by the Rev. J. Croft, Thomas Russell Jarvie, M.R.C.V.S., Gateshead, to Amy Mary Munro, of Novar, Low Fell. At home, Jan. 20th and 21st, 1904.

MR. J. E. RICKARDS, M.R.C.V.S.

It is with the deepest regret we record the death of Mr. J. E. Rickards, M.R.C.V.S., at the early age of 40. He took his diploma in 1884, and in the same year obtained the FitzWygram Prize. As a student he had a distinguished career, and those who were his contemporaries will remember the prominent position he took in all his classes.

On leaving College he commenced practice in Paddington, where his family owned a large horse business, and soon established a reputation as a keen observer and sound practitioner.

Mr. Rickards was too retiring and modest to take the very prominent position as a practitioner that his talents deserved, but those who knew him were always glad to seek his advice and assistance. By his decease the London practitioner has sustained a heavy loss, and veterinary science is the poorer.

He leaves, alas! a large and sorrowing family, to whom the heartfelt sympathy of the profession will be given.

To Fishers.

Under the title of "An Angler's Autobiography," Mr. F. M. Halford has written the fourth and concluding volume of the "Halford Dry Fly Series," which will be published shortly by Messrs. Vinton and Co. The work, which contains twenty-eight full-page photogravures besides many other illustrations, covers the whole field of the author's lengthy experience, and includes chapters on coarse fishing, sea fishing, Thames trouting, salmon fishing, fishing the sunk fly, the test and floating flies, dry-fly fishing, etc. Mr. William Senior has written a highly interesting introduction.

The book will be first issued as an *Edition de Luxe*, limited to 100 copies, numbered and signed, to be followed by the ordinary issue, demy octavo.

CORRESPONDENCE.

"CALORIS."

Sir,

I shall be much obliged if any of your readers who are clinically acquainted with "Caloris," as it is called in Hungary, can give me information regarding it.

From what I have read of this disease I am inclined to think it is similar to if not identical with "Bursattee,' and in my endeavours to establish the etiology of the latter I think I may be assisted by a fuller knowledge of "Caloris," which is no doubt familiar to members residing or travelling in Hungary.—Yours truly,

THE DOCTOR SAHIB.

December 11.

REMARKS ON VETERINARY AND MEDICAL SCIENCE.

Sir,

It was very refreshing to read the discussion on the above subject as reported in *The Veterinary Record* of December 26th. After the experience of the past few days it is satisfactory to learn that in Dublin the practice of "blackmail" by grooms and coachmen is not carried on.

I presume there are degrees of what is known as "blackmail," but if our establishment is invaded from morning to night, at Christmas time, by a crowd of attendants of horses, clamouring for Xmas allowances, and even demanding money, giving abuse if they are refused, or if the allowance is not adequate to their ideas; then indeed the custom must come under the heading of "blackmail" of a very virulent type.

I am quite prepared to be told that this custom should not be carried on, and that it is contrary to the dignity of the profession. When such a remark emanates from a practitioner who is independent and has laid by the "nest egg," I take it for what it is worth, and merely ask him how he manages to satisfy the thirst and greed of these attendants and prevent them from carrying out their dire threats of vengeance. I take it that most veterinary surgeons who keep shoeing forges have bitter experience of the power the attendants of horses possess, and how successfully they punish the practitioner who does not dole out the Xmas allowance. The result is obvious; imaginary faults are found and the loss of a client frequently follows.

Of course if the veterinary surgeon is independent he can afford to fight the matter, but if not he has sad visions of the large weekly wages bill that has to be paid every saturday, and he knows quite well that professional dignity will not pay it. I have heard practitioners boast that they never give tips or Xmas allowances. Further enquiry, however, proved that the opposite was the case, and the man of dignity may be found to "tip" in gold. If he can get his *confrère* to believe that he does not carry out this custom, he gains a point, as naturally the latter will neglect, to a certain extent, the demands of the attendants. *Result*—More clients to Mr. Dignity, while the gullible one is put down as "a wrong 'un" by the attendants, and the owners are told he is careless and ignorant.

It is desirable that this matter should not be cloaked over. I do not mean to infer that the eustom exists everywhere, but I simply give my own experience, and the deductions I have derived therefrom after 17 years work.

The enthusiasm of youth would lead one to believe that scientific knowledge, skill, and ability, with an honest determination to succeed, are the attributes to success in the veterinary profession. A little experience of the world and its ways teaches, however, that success depends to a great extent on the manner with which the attendants of horses regard us. How their goodwill is to be obtained is quickly learned, they care not for soft talk, dignity, or scientific attainments; they are conscious of their power and act accordingly. A few owners of horses may take the trouble to investigate the numerous complaints of a dissatisfied groom or attendant, the majority do not. The new purchase after being passed as sound is constantly giving trouble, and becomes affected with a series of imaginary disorders; perhaps he is reported as off-colour when required, or to shy frequently. The sick horse does not improve as he ought, and Mr. — does not understand the case. The shoeing is badly done, and a shoe falls off in a mysterious manner. Mr. — is not to be found when he is required, so another V.S. has to be called in. These are only a few of the excuses put forward by the groom or attendant who is dissatisfied with his Xmas allowance.

It is often a question with the owner of horses as to which course suits his convenience best, *i.e.*, whether to dismiss the groom or to change his veterinary surgeon. Unfortunately for us the latter course is the one usually adopted.

Having described this evil I shall naturally be asked to suggest a remedy. The most obvious one would be a combination of practitioners to put down the custom. But as there are "black sheep in every fold" it is quite certain that one of this variety, while pretending to act up to his promise, would find ways and means of securing the patronage of the attendants. Judging from the manner in which some stoop to gain practice, the above statement should not cause surprise. It may be that

"The jingle of the guinea
Helps the hurt that honour feels."

Some would suggest to get rid of the forge, and so get out of touch with this class of attendants. My answer is—Another practitioner will start a forge and continue the custom complained of—to his benefit and to my detriment.

Even without a forge, it is customary in a city practice to give Xmas allowances; in fact "tipping" on sundry occasions is expected. No one objects to this custom provided he is not compelled to pay out large sums of money, knowing well the result if he refuses.

In the present day, when we hear so much with reference to the scientific aspect of the profession, and the opportunities it offers to youths of ability and education, it seems hard to point out the dark side of the business part of the calling. The junior who is ignorant of it learns by bitter experience that the path to success is not one of roses; and that the encouraging words of the inaugural lecturer, or the farewell address when leaving College, are by no means directed towards the drawbacks of the profession.

The popular veterinary lecturers have been exonerated from all blame, and even complimented for their labours in the cause of ignorant stockowners who do not wish to pay for the services of veterinary surgeons. Wholesale chemists have been blamed for supplying vaccines and appliances to stockowners, but it would be interesting to enquire who introduced the names of these chemists to these poor (?) ignorant farmers. Who caused the land to be flooded with injections and appliances for the treatment of milk fever, or made the use of black-quarter vaccine a household word? Will anyone seriously maintain that a stockowner having learned how to employ these various remedies, will be so philanthropic to his veterinary surgeon as to pay him to attend and administer them?

But we are told that these lecturers are to be compared to missionaries!!! Well, everyone knows how the Established Churches regard missionaries who come into a district already supplied with churches and clergy. Of course in large cities the popular veterinary lecturer might lecture *ad nauseam* without doing any harm to the practitioners. That the opposite holds good in country districts is known only too well by yours faithfully,

E. Wallis Hoare.

Cork, December, 1903.

ACCIDENT INSURANCE COMPANIES AND VETERINARY SURGEONS.

Sir,

I have read the correspondence on this subject in *The Veterinary Record* with interest. Of course without seeing the policies and the terms thereof it is impossible to give an opinion, but I have never seen one in which a fractured limb was not included, and cannot understand how anyone could accept such a one.

From your correspondent's letter of December 19th I understand that the Company he was insured in offered to submit his claim to arbitration. Of course if there was a clause in the policy to that effect they would be within their rights, but I gather that there was not. If so, then there is at once an admission of the liability of the Company, and it certainly looks like an attempt to avoid their liabilities. In that case had I been the interested party I should without more ado have written to the Secretary requesting payment within a certain date, failing which I should have served the Company a writ. and had the facts of the case been as I suppose from your correspondent's letter, I am certain that before the morning that the case was down for hearing my claim and costs would have been settled out of court.

My experience of insurance companies is that they lean more to discharge doubtful claims than dispute them, for the business is so cut and the competition so great that no company could long last who made a practice of not discharging claims, and, unless certain of winning the case, have great reluctance to appearing in court.—Yours, etc.,

Joshua A. Nunn, Lieut.-Col., Barrister-at-Law.

December 28.

Communications, Books and Papers received.—Messrs. J. B. Wolstenholme, W. A. Dellagana, H. L. Somers, E. W. Hoare, F. D. Coburn (St. Louis). Col. J. A. Nunn, Major H. T. Pease. "Thes."

Annual Report of Imperial Bacteriologist (India) *1902-3*, "*Indirect Advantages of the Motor Car to Horses*," *Yorkshire Post*, *Eastern Daily Express*, *The Onlooker*, *The Caledonian Medical Journal*.